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Last logoff: 30mar09 13:52:23

Logon file405 06apr09 09:20:20

*** ANNOUNCEMENTS ***

*** FREE FILE OF THE MONTH (April) Prompt and Trade & Industry Database (Files 16 and 148)

Each month Dialog offers an opportunity to try out new or unfamiliar sources by offering \$100 of free searching (either DialUnits or connect time) in one specific file. Output and Alerts charges are not included. For more details visit: <http://www.dialog.com/freefile/> and then take a moment to get familiar with another great Dialog resource.

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NEW FILE

***File 651, TRADEMARKSCAN(R) - China. See HELP NEWS 651 for details.

RESUMED UPDATING

***File 523, D&B European Financial Records

RELOADS COMPLETED

***Files 154&155, MEDLINE(R)

***File 126, TRADEMARKSCAN(R) - United Kingdom

***File 228, TRADEMARKSCAN(R) - Spain

***File 672, TRADEMARKSCAN(R) - Germany

***File 655, TRADEMARKSCAN(R) - Korea

***File 656, TRADEMARKSCAN(R) - Australia

***File 657, TRADEMARKSCAN(R) - France

***File 673, TRADEMARKSCAN(R) - Italy

FILES RENAMED

***File 321, PLASPEC now known as Plastic Properties Database

FILES REMOVED

***File 301, CHEMNAME - please use File 398 ChemSearch

***File 388,PEDS: Defense Program Summaries

***File 588,DMS-FI Contract Awards

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* * *

SYSTEM:HOME

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Menu System II: D2 version 1.8.0 term=ASCII

*** DIALOG HOMEBASE(SM) Main Menu ***

Information:

1. Announcements (new files, reloads, etc.)
2. Database, Rates, & Command Descriptions
3. Help in Choosing Databases for Your Topic
4. Customer Services (telephone assistance, training, seminars, etc.)
5. Product Descriptions

Connections:

6. DIALOG(R) Document Delivery
7. Data Star(R)

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/L = Logoff

/NOMENU = Command Mode

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(e.g., B1 for ERIC).

? b 410

06apr09 09:20:20 User226352 Session D1127.1

\$0.00 0.267 DialUnits FileHomeBase

\$0.00 Estimated cost FileHomeBase

\$0.00 Estimated cost this search

\$0.00 Estimated total session cost 0.267 DialUnits

File 410:Dialog Customer Newsletters 2008

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Set	Items	Description
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? set hi ;set hi

HIGHLIGHT set on as ''

HIGHLIGHT set on as ''

? b biochem

06apr09 09:20:26 User226352 Session D1127.2

\$0.00 0.117 DialUnits File410

\$0.00 Estimated cost File410

\$0.02 TELNET

\$0.02 Estimated cost this search

\$0.02 Estimated total session cost 0.384 DialUnits

SYSTEM:OS - DIALOG OneSearch

File 5:Biosis Previews(R) 1926-2009/Mar W5

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File 6:NTIS 1964-2009/Mar W5

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File 24:CSA Life Sciences Abstracts 1966-2009/Jul

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File 34:SciSearch(R) Cited Ref Sci 1990-2009/Mar W5

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*File 40: This file is closed and will no longer update. For similar data, please search File 76-Environmental Sciences.

File 41:Pollution Abstracts 1966-2009/Jul

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File 50:CAB Abstracts 1972-2009/Mar W5

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File 65:Inside Conferences 1993-2009/Apr 03

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File 76:Environmental Sciences 1966-2009/Jul

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File 98:General Sci Abs 1984-2009/Apr

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File 103:Energy SciTec 1974-2009/Mar B1

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*File 103: For access restrictions see Help Restrict.

File 136:BioEngineering Abstracts 1966-2007/Jan
(c) 2007 CSA.

*File 136: This file is closed.

File 143:Biol. & Agric. Index 1983-2009/Mar
(c) 2009 The HW Wilson Co

File 144:Pascal 1973-2009/Apr W1
(c) 2009 INIST/CNRS

File 154:MEDLINE(R) 1990-2009/Apr 02
(c) format only 2009 Dialog

File 155:MEDLINE(R) 1950-2009/Apr 03
(c) format only 2009 Dialog

File 156:ToxFile 1965-2009/Mar W5
(c) format only 2009 Dialog

File 162:Global Health 1983-2009/Mar W5
(c) 2009 CAB International

*File 162: The file has been reloaded and accession numbers have changed. See HELP NEWS 162 for information.

File 172:EMBASE Alert 2009/Apr 03
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File 305:Analytical Abstracts 1980-2009/Mar W1
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*File 305: Alert feature enhanced for multiple files, duplicate removal, customized scheduling. See HELP ALERT.

File 369:New Scientist 1994-2009/Mar W4
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File 370:Science 1996-1999/Jul W3
(c) 1999 AAAS

*File 370: This file is closed (no updates). Use File 47 for more current information.

File 393:Beilstein Database - Abstracts 2008/Q2
(c) 2008 Beilstein GmbH

File 399:CA SEARCH(R) 1967-2009/UD=15015
(c) 2009 American Chemical Society

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IPCR/8 classification codes now searchable as IC=. See HELP NEWSIPCR.

File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
(c) 2006 The Thomson Corp

Set	Items	Description
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? s transmembrane		and domain and (bcma or b cell maturation antigen)
	485220	TRANSMEMBRANE
	2279468	DOMAIN
	1556	BCMA
	124	B CELL MATURATION ANTIGEN
S1	62	TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION		ANTIGEN)
? rd s1		

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.

S2 17 RD S1 (unique items)

? t s2/7/all

>>>Format 7 is not valid in file 143

2/7/1 (Item 1 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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0020026412 BIOSIS NO.: 200800073351

cDNA cloning, expression and bioactivity of porcine BAFF

AUTHOR: Guan Zheng-Bing; Dan Wen-Bing; Shui Yan; Ye Ji-Lin; Zhang
Shuang-Quan (Reprint)

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China

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JOURNAL: Developmental & Comparative Immunology 31 (12): p1211-1219
2007

2007

ITEM IDENTIFIER: doi:10.1016/j.dci.2007.03.006

ISSN: 0145-305X

DOCUMENT TYPE: Article; Editorial

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: B cell activating factor belonging to the tumor necrosis
factor

(TNF) family (BAFF) is critical for B cell survival, maturation and
T

cell activation by acting through its three receptors, BAFF-R, BCMA
and TACI. In the present study, a porcine BAFF cDNA, designated

pBAFF,

was cloned by RT-PCR and rapid amplification of cDNA ends (RACE)
strategies. The full-length cDNA of pBAFF consists of 805 by with a
702

by open reading frame, encoding 233 amino acids. The deduced amino
acid

sequence contains a predicted transmembrane domain and a
putative furin protease cleavage site corresponding to other
identified

BAFF homologues. The amino acid similarity between the functional
soluble

parts of pBAFF and human BAFF (hBAFF) or chicken BAFF (cBAFF) is
93% and

85%, respectively, with identity at the amino acid level was 88%
and 76%,

respectively. The characteristic of the three-cysteine residues of
BAFF

is conserved in pBAFF. RT-PCR showed that BAFF is expressed in many tissues in the pig, including spleen, liver, lung, heart, intestine, kidney, thymus and PBLs. Recombinant soluble pBAFF (psBAFF) fused with

His(6) tag was efficiently expressed in Escherichia coli BL21 (DE3) and

its expression was confirmed by sodium dodecyl sulfate polyacrylamide gel

electrophoresis (SDS-PAGE) and Western blotting. In vitro, purified psBAFF co-stimulates the proliferation of not only porcine B cells but

also human B cells. In addition, hsBAFF binds to porcine B cells and has

a positive effect on their proliferation. These findings indicate pBAFF

plays an important role in proliferation of porcine B cells and functional cross-reactivity occurs between porcine and human BAFF.

In vitro expression of bioactive psBAFF provides the basis for further investigation of its potential to be used as an immunoadjuvant for enhancing vaccine efficacy and an immunotherapeutic in pig. It also provides the basis for investigations on the role of BAFF in this important domestic species and an animal model for human diseases.

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2/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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18631415 BIOSIS NO.: 200510325915
The functional role of DMWD in BAFF-R mediated signal
AUTHOR: Miyazaki Tadaaki (Reprint); Iwai Atsushi; Nakashima Mitsuko; Kimura

Chiemi; Uede Toshimitsu
AUTHOR ADDRESS: Inst Genet Med, Kita Ku, Sapporo, Hokkaido 0600815, Japan**

Japan
JOURNAL: FASEB Journal 19 (4, Suppl. S, Part 1): pA899 MAR 4 2005 2005

CONFERENCE/MEETING: Experimental Biology 2005 Meeting/35th International Congress of Physiological Sciences San Diego, CA, USA March 31 -April 06, 2005; 20050331

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Amer Soc Pharmacol & Expt Therapeut
Int Union Physiol Sci

ISSN: 0892-6638

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: BAFF (B cell activation factor) is a member of the TNF (tumor necrosis factor) ligand family and binds to three receptors, BCMA (B cell maturation antigen), TACI (transmembrane activator and CAML (Calcium-modulating cyclophilin ligand)-interactor) and BAFF-R (BAFF receptor), mostly expressed on mature B lymphocytes. It has been shown that BAFF has a function for B cell proliferation and maturation primarily through BAFF-R. Here, we identified DMWD (dystrophia myotonica containing WD repeat motif, also called DMR-N9) molecule as a critical signal transducer for BAFF-R mediated signaling. We found that DMWD interacts with the cytoplasmic domain of BAFF-R. However, the function of the DMWD gene products is poorly understood. We confirmed the expression of DMWD in human B cells and found that DMWD has a function to regulate interleukin (IL)-10 production mediated through BAFF-R. Furthermore, we evaluated the function of DMWD for NF-kappa B and JNK activation pathways. It was suggested that DMWD has a critical role for these signaling pathways mediated by BAFF-R. In addition, we identified a kinase as a DMWD-interacting protein and analysed the functions of this kinase. We demonstrated that autophosphorylation of this kinase was regulated by DMWD after BAFF stimulation. It was suggested that DMWD regulates the function of this kinase to transduce BAFF-R mediated signaling pathways.

2/7/3 (Item 3 from file: 5)
DIALOG(R) File 5: Biosis Previews(R)
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18396540 BIOSIS NO.: 200510091040
Identification of proteoglycans as the APRIL-specific binding partners
AUTHOR: Ingold Karine; Zumsteg Adrian; Tardivel Aubry; Huard Bertrand;
Steiner Quynh-Giao; Cachero Teresa G; Qiang Fang; Gorelik Leonid;
Kalled
Susan L; Acha-Orbea Hans; Rennert Paul D; Tschopp Juerg; Schneider
Pascal
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JOURNAL: Journal of Experimental Medicine 201 (9): p1375-1383 MAY 2
05

2005

ISSN: 0022-1007

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: B cell activating factor of the tumor necrosis factor (TNF)
family (BAFF) and a proliferation-inducing ligand (APRIL) are closely
related ligands within the TNF superfamily that play important
roles in B

lymphocyte biology. Both ligands share two receptors-transmembrane
activator and calcium signal-modulating cyclophilin ligand
interactor

(TACI) and B cell maturation antigen (BCMA)-that are predominantly
expressed on B cells. In addition, BAFF specifically binds BAFF
receptor,

whereas the nature of a postulated APRIL-specific receptor remains
elusive. We show that the TNF homology domain of APRIL binds
BCMA and TACI, whereas a basic amino acid sequence (QKQKKQ) close
to the NH2 terminus of the mature protein is required for binding
to the

APRIL-specific "receptor." This interactor was identified as
negatively

charged sulfated glycosaminoglycan side chains of proteoglycans.
Although

T cell lines bound little APRIL, the ectopic expression of
glycosaminoglycan-rich syndecans or glypicans conferred on these
cells a

high binding capacity that was completely dependent on APRIL's basic
sequence. Moreover, syndecan-1- positive plasma cells and
proteoglycan-rich nonhematopoietic cells displayed high specific,
heparin-sensitive binding to APRIL. Inhibition of BAFF and APRIL,
but not

BAFF alone, prevented the survival and/or the migration of newly
formed

plasma cells to the bone marrow. In addition, costimulation of B
cell

proliferation by APRIL was only effective upon APRIL
oligomerization.

Therefore, we propose a model whereby APRIL binding to the
extracellular

matrix or to proteoglycan-positive cells induces APRIL
oligomerization,

which is the prerequisite for the triggering of TACI- and/or BCMA
-mediated activation, migration, or survival signals.

2/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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18358730 BIOSIS NO.: 200510053230

Graft-versus-tumor response in patients with multiple myeloma is associated

with antibody response to BCMA, a plasma-cell membrane receptor
AUTHOR: Bellucci Roberto; Alyea Edwin P; Chiaretti Sabina; Wu Catherine J;

Zorn Emmanuel; Weller Edie; Wu Bingyan; Canning Christine; Schlossman

Robert; Munshi Nikhil C; Anderson Kenneth C; Ritz Jerome (Reprint)

AUTHOR ADDRESS: Dana Farber Canc Inst, Dept Med Oncol, 44 Binney St, M530,

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JOURNAL: Blood 105 (10): p3945-3950 MAY 15 05 2005

ISSN: 0006-4971

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: Donor lymphocyte infusions (DLIs) induce effective graft-versus-tumor responses in patients with multiple myeloma who relapse after allogeneic hematopoietic stem-cell transplantation.

The

graft-versus-myeloma response is presumably mediated primarily by donor T

cells, but recent studies have also demonstrated the presence of antibodies specific for a variety of myeloma-associated antigens in patients who achieve complete remission after DLI. One of the B-cell antigens identified in these studies was B-cell maturation antigen (BCMA), a transmembrane receptor of the tumor necrosis factor (TNF) superfamily that is selectively expressed by mature B cells.

The

present studies were undertaken to characterize the functional significance of antibodies to BCMA in vivo. Using transfected cells expressing BCMA, antibodies in patient serum were found to react with the cell-surface domain of BCMA. Post-DLI patient serum was able to induce complement-mediated lysis and antibody-dependent cellular cytotoxicity (ADCC) of transfected cells and primary

myeloma

cells expressing BCMA. BCMA antibodies were only found in post-DLI responders and not in other allogeneic transplant patients

or

healthy donors. These results demonstrate that BCMA is a target of donor B-cell immunity in patients with myeloma who respond to DLI. Antibody responses to cell-surface BCMA may contribute directly to tumor rejection in vivo.

2/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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18274167 BIOSIS NO.: 200500181232
Selectivity of BAFF/BLyS and APRIL for binding to the TNF family
receptors

BAFFR/BR3 and BCMA
AUTHOR: Day Eric S; Cachero Teresa G; Qian Fang; Sun Yaping; Wen
Dingyi;

Pelletier Marc; Hsu Yen-Ming; Whitty Adrian (Reprint)
AUTHOR ADDRESS: Dept Drug DiscoveryCambridge Ctr 14, Biogen Idec Inc,
Cambridge, MA, 02142, USA**USA

AUTHOR E-MAIL ADDRESS: adrian.whitty@biogenidec.com

JOURNAL: Biochemistry 44 (6): p1919-1931 February 15, 2005 2005
MEDIUM: print

ISSN: 0006-2960 _(ISSN print)

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: BAFF (B cell activating factor of the TNF family, also
known as

BlyS and TALL-I), a TNF family cytokine critical for the
development and
function of B cells, has been reported to bind to three receptors,
BCMA (B cell maturation protein), TACI (transmembrane
activator and CAML (calcium-modulator and cyclophilin ligand)
interactor), and BAFFR (BAFF receptor), but with widely conflicting
values for the affinity and selectivity of binding. BCMA and TACI
additionally bind APRIL (a proliferation-inducing ligand), the TNF
family

ligand most homologous to BAFF. Using soluble, monomeric forms of
the

receptors, we demonstrate that BAFFR binds BAFF with KD apprx 16 nM,
while BCMA binds with KD apprx1.6 muM, indicating a apprx100-fold
selectivity for binding to BAFFR over BCMA. APRIL shows the
opposite selectivity, binding to BCMA with KD apprx 16 nM while
showing no detectable affinity for BAFFR (KD > 3 muM). The binding
of

BAFF or APRIL to these receptors is highly sensitive to
assay-dependent

avidity effects, likely explaining the widely ranging affinity
values

reported in the literature. Binding of BAFF to BCMA-Fc, a bivalent
fusion protein consisting of the extracellular domain of BCMA
fused to the hinge and CH1 and CH2 domains of human IgG1, in
solution or

coated onto an ELISA plate gave apparent binding affinities of
apprx0.63

and apprx0.15 nM, respectively, compared to values of KD(app)
ltoreq 30

and approx 100 pM for the corresponding BAFFR/IgG1 fusion protein, BAFFR-Fc. The high selectivity of BAFF for BAFFR versus BCMA is thus partly obscured in these multivalent assays. The intrinsically high selectivity inferred from the measurements with monomeric receptor correlates well with in vivo data from knockout mice, providing a possible explanation for the observations that interruption of the BAFFR gene in the A/WySnJ mouse produces a phenotype similar to the BAFF knockout mouse, while the BCMA knockout mouse has no discernible B cell phenotype.

2/7/6 (Item 6 from file: 5)
DIALOG(R) File 5: Biosis Previews(R)
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17984372 BIOSIS NO.: 200400355161
Methods and compositions of matter concerning APRIL/G70, BCMA, BLYS/AGP-3 and TACI
AUTHOR: Theill Lars Eyde (Reprint); Yu Gang
AUTHOR ADDRESS: Thousand Oaks, CA, USA*USA
JOURNAL: Official Gazette of the United States Patent and Trademark Office
Patents 1285 (2): Aug. 10, 2004 2004
MEDIUM: e-file
PATENT NUMBER: US 6774106 PATENT DATE GRANTED: August 10, 2004
20040810
PATENT CLASSIFICATION: 514-12 PATENT ASSIGNEE: Amgen Inc.
PATENT COUNTRY: USA
ISSN: 0098-1133 _(ISSN print)
DOCUMENT TYPE: Patent
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This invention concerns interactions among APRIL/G70, AGP-3/BLYS, BCMA, and TACI and related methods of use and compositions of matter. It has been found that (1) sAPRIL/G70 binds to the cell-surface receptors BCMA and TACI on T and B lymphoma cells, resulting in stimulation of proliferation of primary human and mouse B and T cells both in vitro and in vivo; (2) APRIL competes with AGP3's binding to TACI and BCMA; (3) sBCMA inhibits APRIL and AGP3 binding to its receptors; (4) sBCMA ameliorates T cell dependent and T cell independent humoral immune responses in vivo; (5) sTACI inhibits APRIL and AGP3 binding to its receptors and ameliorates T cell dependent and T cell independent humoral immune responses in vivo; and (6) BCMA exhibits similarity with TACI within a single cysteine rich domain located

N-terminal to a potential transmembrane domain. These discoveries provides a strategy for development of therapeutics for treatment of autoimmune diseases, and cancer, for prevention of transplant rejection. Disease states and disease parameters associated with APRIL and AGP-3 may be affected by modulation of BCMA or TACI; disease states and parameters associated with TACI can be affected by modulation of APRIL; disease states and parameters can be affected by modulation of any of TACI, BCMA, APRIL and AGP-3 by a single therapeutic agent or two or more therapeutic agents together.

2/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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17732488 BIOSIS NO.: 200400103245
Chicken BAFF: A highly conserved cytokine that mediates B cell survival.
AUTHOR: Schneider Kirsten; Kothlow Sonja; Schnelder Pascal; Tardivel Aubry;
Goebel Thomas; Kaspers Bernd; Staeheli Peter (Reprint)
AUTHOR ADDRESS: Abteilung Virologie, Institut fuer Medizinische Mikrobiologie und Hygiene, University of Freiburg, 79104, Freiburg, Germany**Germany
AUTHOR E-MAIL ADDRESS: staeheli@ukl.uni-freiburg.de
JOURNAL: International Immunology 16 (1): p139-148 January 2004 2004
MEDIUM: print
ISSN: 0953-8178
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Members of the tumor necrosis factor (TNF) family play key roles in the regulation of inflammation, immune responses and tissue homeostasis. Here we describe the identification of the chicken homologue of mammalian B cell activating factor of the TNF family (BAFF/BLyS). By searching a chicken EST database we identified two overlapping cDNA clones that code for the entire open reading frame of chicken BAFF (chBAFF), which contains a predicted transmembrane domain and a putative furin protease cleavage site like its mammalian counterparts. The amino acid identity between soluble chicken and human BAFF is 76%, considerably higher than for most other known cytokines. The chBAFF gene is most strongly expressed in the bursa of Fabricius. Soluble recombinant

chBAFF produced by human 293T cells interacted with the mammalian cell-surface receptors TACI, BCMA and BAFF-R. It bound to chicken B cells, but not to other lymphocytes, and it promoted the survival of splenic chicken B cells in culture. Furthermore, bacterially expressed chBAFF induced the selective expansion of B cells in the spleen and cecal tonsils when administered to young chicks. Our results suggest that like its mammalian counterpart, chBAFF plays an important role in survival and/or proliferation of chicken B cells.

2/7/8 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

18203451 Genuine Article#: 338A0 Number of References: 24
Title: A novel bioassay for B-cell activating factor (BAFF) based on expression of a BAFF-receptor ectodomain-tumour necrosis factor-related apoptosis-inducing ligand (TRAIL) receptor-2 endodomain fusion receptor in human rhabdomyosarcoma cells
Author(s): McClements M; Williams S; Ball C; Bristow A; Wadhwa M; Meager A
(REPRINT)
Corporate Source: Natl Inst Biol Stand & Controls, Blanche Lane, Cytokine & Growth Factor Sect, Biotherapeut Grp, S Mimms EN6 3QG/Herts/England/
(REPRINT); Natl Inst Biol Stand & Controls, Blanche Lane, Cytokine & Growth Factor Sect, Biotherapeut Grp, S Mimms EN6 3QG/Herts/England/;
Natl Inst Biol Stand & Controls, Blanche Lane, Prot Sci Sect, Technol Dev & Infrastruct Grp, S Mimms EN6 3QG/Herts/England/
Journal: JOURNAL OF IMMUNOLOGICAL METHODS, 2008, V337, N1 (AUG 20), P63-70
ISSN: 0022-1759 Publication date: 20080820
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS
Language: English Document Type: ARTICLE
Abstract: B-cell activating factor (BAFF) is a type II transmembrane glycoprotein belonging to the tumour necrosis factor ligand superfamily. Active soluble forms of BAFF are generated either by cleavage of the extracellular domain or by recombinant DNA technology. The current bioassay for measuring the activity of soluble BAFF involves stimulation of the proliferation of mouse splenic B-cells

in the presence of goat anti-mouse IgM mu chain which is rather cumbersome and lengthy and yields variable results. We have therefore developed an alternative functional assay which relies on the ability of BAFF to induce an apoptotic response in human rhabdomyosarcoma cells. For this, we constructed a chimeric receptor containing the ectodomain of the MuBAFF-R - the major cell receptor for BAFF - and the endodomain of the HuTRAIL-R2 - one of the two functional receptors for TRAIL - which is known to contain a death domain and trigger apoptosis. When the chimeric receptor was expressed in the TRAIL-sensitive human rhabdomyosarcoma cell line KD4 clone 21, recombinant BAFF of either human or mouse sequence stimulated apoptosis, similar to TRAIL, in a dose-dependent manner. The transfected cell population, called FL17, expressing the MuBAFF-R/HuTRAIL-R2 thus provided the basis of a novel functional bioassay for BAFF that is simple and relatively fast to perform. The construction of the chimeric receptor, development of the transfected cells expressing this receptor and the development of sensitive and reproducible bioassays for BAFF and anti-BAFF neutralising antibodies are described.

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2/7/9 (Item 2 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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09831500 Genuine Article#: 453UW Number of References: 20
Title: The role of TALL-1 and APRIL in immune regulation
Author(s): Khare SD (REPRINT) ; Hsu HL
Corporate Source: Amgen Inc, Dept Pathol Pharmacol, 1 Amgen Ctr Dr/Thousand Oaks//CA/91320 (REPRINT); Amgen Inc, Dept Pathol Pharmacol, Thousand Oaks//CA/91320; Amgen Inc, Dept Inflammation, Thousand Oaks//CA/91320
Journal: TRENDS IN IMMUNOLOGY, 2001, V22, N2 (FEB), P61-63
ISSN: 1471-4906 Publication date: 20010200
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND
Language: English Document Type: EDITORIAL MATERIAL
Abstract: Members of the tumor necrosis factor (TNF) superfamily play important roles in cell proliferation and death during immune regulation. Most members are synthesized as type II transmembrane proteins; the carboxy terminal extracellular domain can be cleaved from the cell membrane to form soluble active cytokines that bind to appropriate members of the TNF receptor family. Here, we

describe the biological significance of recently discovered members of the TNF superfamily (TALL-1 and APRIL) and their receptors (TACI and BCMA) in the pathophysiology of human diseases.

2/7/10 (Item 3 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
(c) 2009 The Thomson Corp. All rts. reserv.

07240275 Genuine Article#: 140DX Number of References: 59
Title: The characterization of murine BCMA gene defines it as a new member of the tumor necrosis factor receptor superfamily
Author(s): Madry C; Laabi Y; Callebaut I; Roussel J; Hatzoglou A; LeConiat M; Mornon JP; Berger R; Tsapis A (REPRINT)
Corporate Source: UNIV PARIS 11,INSERM CJF 95 02, CTR RECH, FAC MED PARIS
SUD, 32 RUE CARNETS/F-92140 CLAMART//FRANCE/ (REPRINT); UNIV PARIS 11,INSERM CJF 95 02, CTR RECH, FAC MED PARIS SUD/F-92140 CLAMART//FRANCE/; UNIV PARIS 06,CNRS UMR C7590, LAB MINERAL CRISTALLOG/F-75005 PARIS//FRANCE/; INST GENET MOL,INSERM U301/F-75010 PARIS//FRANCE/
Journal: INTERNATIONAL IMMUNOLOGY, 1998, V10, N11 (NOV), P1693-1702
ISSN: 0953-8178 Publication date: 19981100
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD OX2 6DP, ENGLAND
Language: English Document Type: ARTICLE
Abstract: The BCMA gene is a new gene discovered by the molecular analysis of a t(4;16) translocation, characteristic of a human T cell lymphoma. It has no significant similarity with any known protein or motif, so that its function was unknown. This report describes the cloning of murine BCMA cDNA and its genomic counterpart. The mouse gene is organized into three exons, like the human gene, and lies in murine chromosome 16, in the 16B3 band, the counterpart of the human chromosome 16p13 band, where the human gene lies. Murine BCMA cDNA encodes a 185 amino acids protein (184 residues for the human), has a potential central transmembrane segment like the human protein and is 62% identical to it. The murine BCMA mRNA is found mainly in lymphoid tissues, as is human BCMA mRNA, Alignment of the murine and human BCMA protein sequences revealed a conserved motif of six cysteines in the N-terminal part, which strongly suggests that the BCMA protein belongs to the tumor necrosis factor receptor (TNFR) superfamily, Human BCMA is the first member of

the TNFR family to be implicated in a chromosomal translocation.

2/7/11 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE
(c) 2009 Elsevier B.V. All rts. reserv.

0005585389 SUPPLIER NUMBER: 2004081899
TWE-PRIL; a fusion protein of TWEAK and APRIL
Kolfchoten G.M.; Pradet-Balade B.; Hahne M.; Medema J.P.
AUTHOR EMAIL: j.p.medema@lumc.nl
CORRESP. AUTHOR/AFFIL: Medema J.P., Department of Clinical Oncology,
Leiden
University Medical Center, Albinusdreef 2a, 2333 ZA Leiden,
Netherlands
CORRESP. AUTHOR EMAIL: j.p.medema@lumc.nl
Journal: Biochemical Pharmacology (Biochem. Pharmacol.), v66, n8,
(1427-1432), 2003, United States
PUBLICATION DATE: October 15, 2003 (20031015)
CODEN: BCPCA
ISSN: 0006-2952 eISSN: 1471-2970
DOI: [http://dx.doi.org/10.1016/S0006-2952\(03\)00493-3](http://dx.doi.org/10.1016/S0006-2952(03)00493-3)
PUBLISHER ITEM IDENTIFIER: S0006295203004933
RECORD TYPE: Abstract; New
DOCUMENT TYPE: Conference Paper
LANGUAGES: English SUMMARY LANGUAGES: English
NO. OF REFERENCES: 61

TWEAK and APRIL are both members of the tumor necrosis factor family, which are involved in respectively angiogenesis and immune regulation. While TWEAK is processed at the cell surface, APRIL is processed inside the cell by a furin-convertase and is solely able to perform its function as a soluble factor. Recently, TWE-PRIL has been identified, which is an endogenous hybrid transcript between TWEAK and APRIL. TWE-PRIL is a transmembrane protein that consists of a TWEAK intracellular, transmembrane and stalk region combined with APRIL as its receptor-binding domain. As such TWE-PRIL is expressed at the cell surface. Although TWE-PRIL, like APRIL, can stimulate T and B cell lines, distinct biological functions that may result from its membrane anchoring cannot be excluded. Understanding the function of this newly identified protein will contribute to the elucidation of the complexity of the tumor necrosis factor family. (c) 2003 Elsevier Inc. All rights reserved.

2/7/12 (Item 1 from file: 72)
DIALOG(R)File 72:EMBASE
(c) 2009 Elsevier B.V. All rts. reserv.

0082845246 EMBASE No: 2009045695
Physiological roles and mechanisms of signaling by TRAF2 and TRAF5
ISSUE TITLE: TNF Receptor Associated Factors (TRAFs)
Au P.-Y.B.; Yeh W.-C.
University Health Network, Department of Medical Biophysics,
University
of Toronto, Toronto, ON, Canada
AUTHOR EMAIL: wyeh@uhnres.utoronto.ca
CORRESP. AUTHOR/AFFIL: Yeh W.-C.: University Health Network,
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of Medical Biophysics, University of Toronto, Toronto, ON M5G 2C1,
Canada
CORRESP. AUTHOR EMAIL: wyeh@uhnres.utoronto.ca
EDITOR(S): Wu H.
Weill Medical College of Cornell Uni, New York, NY, United States

Advances in Experimental Medicine and Biology (Adv. Exp. Med.
Biol.) (

United States) December 1, 2007, 597/- (32-47)
CODEN: AEMBA ISSN: 0065-2598 ISBN: 9780387706290
DOI: 10.1007/978-0-387-70630-6-3
DOCUMENT TYPE: Book Series; Review RECORD TYPE: Abstract
LANGUAGE: English SUMMARY LANGUAGE: English
NUMBER OF REFERENCES: 126

TRAF2 and TRAF5 are closely related members of the TRAF family of
proteins. They are important signal transducers for a wide range of
TNF
receptor superfamily members, including TNFR1, TNFR2, CD40 and other
lymphocyte costimulatory receptors, RANK/TRANCE-R, EDAR, LTbetaR,
LMP-1 and
IRE1. TRAF2 and TRAF5 therefore regulate diverse physiological roles,
ranging from T and B cell signaling and inflammatory responses to
organogenesis and cell survival. The major pathways mediated by TRAF2
and
TRAF5 are the classical and alternative pathways of NF-kappaB
activation,
and MAPK and JNK activation. TRAF2 is heavily regulated by ubiquitin
signals, and many of the signaling functions of TRAF2 are mediated
through
its RING domain and likely its own role as an E3 ubiquitin ligase.
(c) 2007 Landes Bioscience and Springer Science+Business Media, LLC.

2/7/13 (Item 2 from file: 72)
DIALOG(R)File 72:EMBASE
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0080455577 EMBASE No: 2005099733

TNF receptor (TNFR)-associated factor (TRAF) 3 serves as an inhibitor of TRAF2/5-mediated activation of the noncanonical NF-kappaB pathway by TRAF-binding TNFRs

Hauer J.; Puschner S.; Ramakrishnan P.; Simon U.; Bongers M.; Federle C.; Engelmann H.

Institut für Immunologie, Universität München, Goethestrasse 31, 80366

Munich, Germany

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Proceedings of the National Academy of Sciences of the United States of

America (Proc. Natl. Acad. Sci. U. S. A.) (United States)

February 22,

2005, 102/8 (2874-2879)

CODEN: PNAS ISSN: 0027-8424

DOI: 10.1073/pnas.0500187102

DOCUMENT TYPE: Journal; Article RECORD TYPE: Abstract

LANGUAGE: English SUMMARY LANGUAGE: English

NUMBER OF REFERENCES: 46

TNF family members and their receptors contribute to increased gene expression for inflammatory processes and intracellular cascades leading to programmed cell death, both via activation of NF-kappaB. TNF receptor (TNFR)-associated factors (TRAFs) are cytoplasmic adaptor proteins binding

to various receptors of the TNFR family. In an attempt to delineate the

role of individual TRAFs, we compared NF-kappaB activation by CD40 SUB wt

and CD40 mutants with different TRAF recruitment patterns. Recognized only

recently, NF-kappaB signaling occurs at least via two different pathways.

Each pathway results in nuclear translocation of two different Rel-dimers,

the canonical p50/RelA and the noncanonical p52/RelB. Here, we show that

via TRAF6, CD40 mediates only the activation of the canonical NF-kappaB

pathway. Via TRAF2/5, CD40 activates both the canonical and the noncanonical NF-kappaB pathways. We observed that TRAF3 specifically blocked the NF-kappaB activation via TRAF2/5. This inhibitory effect of

TRAF3 depends on the presence of an intact zinc finger domain.

Paradoxically, suppression of TRAF2/5-mediated NF-kappaB activation by TRAF3 resulted in enhanced transcriptional activity of TRAF3-mediated canonical NF-kappaB emanating from CD40. We also observed that 12 TNFR family members (p75TNFR, LTbetaR, RANK, HVEM, CD40, CD30, CD27, 4-1BB, GITR, BCMA, OX40, and TAC1) are each capable of activating the alternative NF-kappaB pathway and conclude that TRAF3 serves as a negative regulator of this pathway for all tested receptors. (c) 2005 by The National Academy of Sciences of the USA.

2/7/14 (Item 1 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

28253583 PMID: 16572839
[Cloning, soluble expression and characterization of human sBCMA]
Guan Zheng-Bing; Cao Peng; Ye Ji-Lin; Zhang Shuang-Quan
Jiangsu Province Key Laboratory for Molecular and Medical
Biotechnology,
Life Sciences College, Nanjing Normal University, Nanjing 210097,
China.

Sheng wu gong cheng xue bao = Chinese journal of biotechnology
(China)
Jan 2006, 22 (1) p46-51, ISSN 1000-3061--Print Journal Code:
9426463

Publishing Model Print
Document type: English Abstract; Journal Article; Research
Support,
Non-U.S. Gov't

Languages: CHINESE
Main Citation Owner: NLM
Record type: In Process

BCMA is one of the transmembrane receptors belonging to BAFF
and APRIL. In order to identify the feasibility of sBCMA as decoy
receptor
and obtain active sBCMA for its structural and functional
research, full
length of hBCMA was amplified with total RNA from Raji cell line by
RT-PCR,
and the cDNA encoding the extracellular soluble domain of hBCMA was
inserted into pET43.1a(+) vector. The recombinant vector
pET43.1a(+)-sBCMA
was transformed into E. coli Origami B(DE3) pLYS which is
helpful for
disulfide bond construction of expression proteins. After IPTG
induction,
the recombinant protein was expressed as soluble fusion
protein,
sBCMA-NusA-His6, and identified by western blotting. Then the
target
protein was purified by Ni(+)-chelating Sepharose Fast Flow. The
binding

activity between recombinant sBCMA and BAFF was detected by ELISA. Also, Recombinant sBCMA inhibited proliferation of mouse B cell stimulating by rhsBAFF. It was proved that recombinant sBCMA has good bioactivity and the method to express those proteins rich in disulfide bond is feasible and effectual.

Record Date Created: 20060331

2/7/15 (Item 2 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

16421452 PMID: 15542592

Structures of APRIL-receptor complexes: like BCMA, TACI employs only a single cysteine-rich domain for high affinity ligand binding.

Hymowitz Sarah G; Patel Darshana R; Wallweber Heidi J A; Runyon Steven;
Yan Minhong; Yin Jianping; Shriver Stephanie K; Gordon Nathaniel C; Pan

Borlan; Skelton Nicholas J; Kelley Robert F; Starovasnik Melissa A
Department of Protein Engineering, Molecular Oncology, Medicinal Chemistry, and Immunology, Genentech, Inc., South San Francisco, California
94080, USA.

Journal of biological chemistry (United States) Feb 25 2005, 280 (8)

p7218-27, ISSN 0021-9258--Print Journal Code: 2985121R

Publishing Model Print-Electronic

Document type: Journal Article; Research Support, U.S. Gov't, Non-P.H.S.

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

TACI is a member of the tumor necrosis factor receptor superfamily and serves as a key regulator of B cell function. TACI binds two ligands, APRIL and BAFF, with high affinity and contains two cysteine-rich domains (CRDs) in its extracellular region; in contrast, BCMA and BR3, the other known high affinity receptors for APRIL and BAFF, respectively, contain only a single or partial CRD. However, another form of TACI exists wherein the N-terminal CRD is removed by alternative splicing. We find that this shorter form is capable of ligand-induced cell signaling and that the

second CRD alone (TACI d2) contains full affinity for both ligands. Furthermore, we report the solution structure and alanine-scanning mutagenesis of TACI d2 along with co-crystal structures of APRIL.TACI d2 and APRIL.BCMA complexes that together reveal the mechanism by which TACI engages high affinity ligand binding through a single CRD, and we highlight sources of ligand-receptor specificity within the APRIL/BAFF system.

Record Date Created: 20050221

Record Date Completed: 20050408

Date of Electronic Publication: 20041112

2/7/16 (Item 3 from file: 154)
DIALOG(R)File 154:MEDLINE(R)
(c) format only 2009 Dialog. All rts. reserv.

15230933 PMID: 12594954

Loss of TACI causes fatal lymphoproliferation and autoimmunity, establishing TACI as an inhibitory BlyS receptor.

Seshasayee Dhaya; Valdez Patricia; Yan Minhong; Dixit Vishva M; Tumas Daniel; Grewal Iqbal S

Department of Immunology, Genentech, Inc, 1 DNA Way, South San Francisco, CA 94080, USA.

Immunity (United States) Feb 2003, 18 (2) p279-88, ISSN 1074-7613--

Print Journal Code: 9432918

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

BlyS, a key cytokine that sustains B cell maturation and tolerance, binds three receptors: BR3, BCMA, and TACI. Results from knockout mice implicate a major functional role for BR3 and a redundant one for BCMA in B cell function. TACI's role is controversial based on defects in TI antibody responses accompanied by B cell hyperplasia in knockout mice. We have presently characterized a precise role for TACI in vivo. TACI(-/-) mice develop fatal autoimmune glomerulonephritis, proteinurea, and elevated levels of circulating autoantibodies. Treatment

of B cells with TACI agonistic antibodies inhibits proliferation in vitro and activation of a chimeric receptor containing the TACI intracellular domain induces apoptosis. These results demonstrate the critical requirement for TACI in regulating B cell homeostasis.

Record Date Created: 20030221

Record Date Completed: 20030325

2/7/17 (Item 1 from file: 399)

DIALOG(R)File 399:CA SEARCH(R)

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133103739 CA: 133(8)103739s PATENT

Soluble receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in

modulating the

immune response and treating autoimmune disorders

INVENTOR(AUTHOR): Gross, Jane A.; Xu, Wenfeng; Madden, Karen; Yee,

David

P.

LOCATION: USA

ASSIGNEE: Zymogenetics, Inc.

PATENT: PCT International ; WO 200040716 A2 DATE: 20000713

APPLICATION: WO 2000US396 (20000107) *US 226533 (19990107)

PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English

PATENT CLASSIFICATIONS:

CLASS: C12N-015/11A; C07K-014/705B; A61K-038/17B; A61K-039/395B

DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY;

CA; CH;

CN; CR; CU; CZ; DE; DK; DM; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU;

ID; IL;

IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG;

MK; MN;

MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR;

TT; UA;

UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM

DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW;

AT; BE;

CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF;

BJ; CF;

CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

SECTION:

CA215010 Immunochemistry

IDENTIFIERS: TACI isoform BR43x2 immune modulation autoimmune disorder,

sequence BR43x2 protein cDNA human mouse, BCMA protein immune modulation autoimmune disorder

DESCRIPTORS:

Proteins,specific or class...

BCMA (B cell membrane antigen); sol. receptor BR43x2 isoform of

transmembrane activator and CAML-interactor TACI and related proteins
and their use in modulating the immune response and treating autoi
Bronchi...
bronchitis, treatment of; sol. receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune
Proteins,specific or class...
BR43x2; sol. receptor BR43x2 isoform of transmembrane activator
and
CAML-interactor TACI and related proteins and their use in
modulating
the immune response and treating autoimmune disorders
Proteins,specific or class...
CAML (calcium-modulator and cyclophilin ligand); sol. receptor
BR43x2
isoform of transmembrane activator and CAML-interactor TACI and
related
proteins and their use in modulating the immune response a
Intestine...
colon, expression specificity in; sol. receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating aut
Intestine,disease...
Crohn's, treatment of; sol. receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Joint,anatomical...
disease, treatment of; sol. receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Appendix... B cell(lymphocyte)... Bone marrow... Lung... Lymph node...
Lymphoma... Salivary gland... Spleen... Stomach... Testis...
Trachea(anatomical)...
expression specificity in; sol. receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating
autoimmune
Immunoglobulins...
fusion products; sol. receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune
disorders

Transplant and Transplantation...

graft-vs.-host reaction, treatment of; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating autoimmune disorders...

Antibodies... humanized; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating

the immune response and treating autoimmune disorders

Diabetes mellitus...

insulin-dependent, treatment of; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating autoimmune disorders... Tumor necrosis factors...

ligand neutrokinine α ; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating autoimmune disorders

Antibodies...

monoclonal; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in

modulating the immune response and treating autoimmune disorders

Nerve,disease...

neuropathy, Ig light chain, treatment of; sol. receptor BR43x2 isoform

of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders

Cytokines...

neutrokinine α ; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use

in modulating the immune response and treating autoimmune disorders... Lymphoma...

non-Hodgkin's, expression specificity in; sol. receptor BR43x2 isoform

of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders... Salivary gland...

parotid, neoplasm, expression specificity in; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related

proteins and their use in modulating the immune response and treating autoimmune disorders... Animal cell line...

Raji, expression specificity in; sol. receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related

proteins

and their use in modulating the immune response and treating auto
 Shock(circulatory collapse)...
 septic, treatment of; sol. receptor BR43x2 isoform of
 transmembrane
 activator and CAML-interactor TACI and related proteins and their
 use
 in modulating the immune response and treating autoimmune diso
 Intestine...
 small, expression specificity in; sol. receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating aut
 Antibodies... cDNA sequences... Drugs... Gene therapy...
 Immunosuppression
 ... Mammal(Mammalia)... Molecular cloning... Mouse... Primate...
 Protein
 sequences...
 sol. receptor BR43x2 isoform of transmembrane activator and
 CAML-interactor TACI and related proteins and their use in
 modulating
 the immune response and treating autoimmune disorders
 Lupus erythematosus...
 systemic, treatment of; sol. receptor BR43x2 isoform of
 transmembrane
 activator and CAML-interactor TACI and related proteins and their
 use
 in modulating the immune response and treating autoimmune di
 Proteins,specific or class...
 TACI (transmembrane activator and CAML-interactor); sol. receptor
 BR43x2 isoform of transmembrane activator and CAML-interactor
 TACI and
 related proteins and their use in modulating the immune respons
 Amyloidosis... Anemia(disease)... Asthma... Autoimmune disease...
 Emphysema
 ... Inflammation... Kidney,disease... Kidney,neoplasm... Multiple
 myeloma
 ... Multiple sclerosis... Myasthenia gravis... Rheumatoid arthritis...
 Swelling,biological... Transplant rejection...
 treatment of; sol. receptor BR43x2 isoform of transmembrane
 activator
 and CAML-interactor TACI and related proteins and their use in
 modulating the immune response and treating autoimmune disorders
 Fusion proteins(chimeric proteins)...
 with Ig heavy chain; sol. receptor BR43x2 isoform of transmembrane
 activator and CAML-interactor TACI and related proteins and their
 use
 in modulating the immune response and treating autoimmune disor
 CAS REGISTRY NUMBERS:
 217638-65-8 282738-49-2 amino acid sequence; sol. receptor BR43x2
 isoform
 of transmembrane activator and CAML-interactor TACI and related
 proteins and their use in modulating the immune response and
 treating

autoimmune disorders
 283157-99-3 cysteine-rich pseudo repeat domain; sol. receptor BR43x2
 isoform of transmembrane activator and CAML-interactor TACI and
 related
 proteins and their use in modulating the immune response and
 treating
 autoimmune disorders
 156253-82-6 198123-04-5 282738-43-6 282738-45-8 282738-47-0
 282738-48-1 nucleotide sequence; sol. receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 148997-64-2D 198029-64-0D 282738-44-7D 282738-46-9D subfragments
 are
 claimed, amino acid sequence; sol. receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 225456-59-7 271755-95-4 274950-83-3 274950-84-4 282738-77-6
 282738-78-7 282738-79-8 282738-80-1 282738-81-2 282738-82-3
 282738-83-4 282738-84-5 282738-85-6 282738-86-7 282738-87-8
 282738-88-9 282738-89-0 282738-90-3 282738-91-4 282738-92-5
 282738-93-6 282738-94-7 282738-95-8 282738-96-9 282738-97-0
 282738-98-1 282738-99-2 282739-00-8 282739-02-0 282739-03-1
 282739-04-2 282739-05-3 282739-06-4 282739-07-5 282739-08-6
 282739-09-7 282739-10-0 282739-11-1 282739-12-2 282739-13-3
 unclaimed nucleotide sequence; sol. receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 282738-76-5 282739-01-9 unclaimed protein sequence; sol. receptor
 BR43x2
 isoform of transmembrane activator and CAML-interactor TACI and
 related
 proteins and their use in modulating the immune response and
 treating
 autoimmune disorders
 98849-88-8 256922-04-0 282729-02-6 282729-03-7 unclaimed
 sequence; sol.
 receptor BR43x2 isoform of transmembrane activator and
 CAML-interactor
 TACI and related proteins and their use in modulating the immune
 response and treating autoimmune disorders
 ? ds

Set	Items	Description
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S1          62    TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION AN-
              TIGEN)
S2          17    RD S1 (unique items)
? ds

Set      Items  Description
S1       62     TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION AN-
              TIGEN)
S2       17     RD S1 (unique items)
? s BCMA or (b(w) cell(w) maturation (w) antigen)
Processing
Processed 10 of 29 files ...
Processing
Processing
Processed 20 of 29 files ...
Processing
Completed processing all files
          1556 BCMA
          9559397 B
          22153748 CELL
          779077 MATURATION
          3353699 ANTIGEN
           643 B(W) CELL(W) MATURATION(W) ANTIGEN
      S3     1744 BCMA OR (B(W) CELL(W) MATURATION (W) ANTIGEN)
? s soluble and s3
          1456508 SOLUBLE
           1744 S3
      S4     280 SOLUBLE AND S3
? rd s4

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
      S5       73 RD S4 (unique items)
? s s5 not PY>2001
Processing
Processed 10 of 29 files ...
Processing
Processed 20 of 29 files ...
Completed processing all files
          73 S5
          54517690 PY>2001
      S6       13 S5 NOT PY>2001
? t s6/7/all
>>>Format 7 is not valid in file 143

6/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16667984 BIOSIS NO.: 200200261495

Therapeutic potential of antagonizing BLYS for chronic lymphocytic leukemia

AUTHOR: Zhou Tong (Reprint); Liu Weimin (Reprint); Zhao Limin (Reprint);

Carter Robert H (Reprint); Kimberly Robert P (Reprint); Emanuel Peter D

AUTHOR ADDRESS: Division of Clinical Immunology and Rheumatology, University of Alabama at Birmingham, Birmingham, AL, USA**USA

JOURNAL: Blood 98 (11 Part 1): p808a November 16, 2001 2001

MEDIUM: print

CONFERENCE/MEETING: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001; 20011207

SPONSOR: American Society of Hematology

ISSN: 0006-4971

DOCUMENT TYPE: Meeting; Meeting Abstract

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The pathogenesis of the accumulation of malignant B cells with a

mature phenotype in CLL is poorly understood, and curative therapies for

CLL do not yet exist. B Lymphocyte Stimulator (BLYS) is a newly identified B cell survival factor of the TNF superfamily, which plays a

crucial role in B cell development and autoimmune disease. At least three

receptors for BLYS have been identified: TACI, BCMA and BAFF-R. To determine the role of BLYS and its receptors in CLL, we examined expression of two receptors: TACI and BCMA using newly generated monoclonal antibodies. Expression of cell surface BLYS with TACI-Fc fusion protein; and expression of soluble BLYS by ELISA was examined in primary peripheral blood cells from 11 patients with CLL.

Similar to normal B cells, the B cells from all CLL patients expressed

high levels of BLYS binding receptors. However, while normal B cells did

not express significant levels of either TACI or BCMA, the CLL B cells from >50% (6/11) and 80% (9/11) patients expressed increased levels

of TACI and BCMA, respectively, indicating that expression of TACI and BCMA is upregulated in CLL. Normal B cells did not express cell surface BLYS as detected by TACI-Fc fusion protein. In contrast, the CLL

B cells from >80% (9/11) had higher levels of cell surface BLYS, which

was confirmed by RT-PCR and Western blot analysis using BLYS specific

primers and antibody. These results suggest that a positive autocrine

loop through cell surface BlyS and its receptors might play a crucial role in the survival and accumulation of CLL B cells. Surprisingly, serum levels of BlyS were almost undetectable in all patients with CLL, suggesting that the circulating BlyS might be over-consumed by the CLL B cells. Compared to normal B cells, the CLL B cells had a poorer response to stimulation with exogenous BlyS in vitro, suggesting that endogenous BlyS is sufficient to sustain the survival of CLL B cells. However, in vitro treatment with TACI-Fc and a BlyS neutralizing monoclonal antibody (15c10) resulted in decreased survival of the CLL B cells in a time-dependent fashion. To further determine the therapeutic potential of blocking BlyS, NK-depleted NOD/SCID mice were reconstituted with the CLL B cells and treated with three doses of 100µg TACI-Fc. Seven days after transfer, the CLL B cells were recovered from the spleens of the recipient mice. The CLL B cells from all 11 patients exhibited the ability to repopulate in the spleens of untreated SCID mice as determined by CD19+ cell number. Treatment with isotype control IgG1 did not alter the cell number recovered from the spleen. However, the number of CLL B cells isolated from majority of patients (8/11) was significantly reduced (50%-85% reduction) in the spleens of recipient mice treated with TACI-Fc. Taken together, our results indicate that BlyS and its receptors, TACI and BCMA, play a crucial role in the survival and accumulation of malignant B cells in CLL patients. Thus, blockade of BlyS stimulation with TACI-Fc or BlyS neutralizing antibody may prove to be an effective treatment for CLL.

6/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16459180 BIOSIS NO.: 200200052691
Maturation of marginal zone and follicular B cells requires B cell activating factor of the tumor necrosis factor family and is independent of B cell maturation antigen
AUTHOR: Schneider Pascal; Takatsuka Hisakazu; Wilson Anne; MacKay Fabienne;

Tardivel Aubry; Lens Susanne; Cachero Teresa G; Finke Daniela;
Beermann
Friedrich; Tschopp Jurg (Reprint)
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Ch. des
Boveresses 155, CH-1066, Epalinges, Switzerland**Switzerland
JOURNAL: Journal of Experimental Medicine 194 (11): p1691-1697
December 3,
2001 2001
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: B cells undergo a complex series of maturation and selection steps in the bone marrow and spleen during differentiation into mature immune effector cells. The tumor necrosis factor (TNF) family member B cell activating factor of the TNF family (BAFF) (BLyS/TALL-1) plays an important role in B cell homeostasis. BAFF and its close homologue a proliferation-inducing ligand (APRIL) have both been shown to interact with at least two receptors, B cell maturation antigen (BCMA) and transmembrane activator and cyclophilin ligand interactor (TACI), however their relative contribution in transducing BAFF signals in vivo remains unclear. To functionally inactivate both BAFF and APRIL, mice transgenic for a soluble form of TACI were generated. They display a developmental block of B cell maturation in the periphery, leading to a severe depletion of marginal zone and follicular B2 B cells, but not of peritoneal B1 B cells. In contrast, mice transgenic for a soluble form of BCMA, which binds APRIL, have no detectable B cell phenotype. This demonstrates a crucial role for BAFF in B cell maturation and strongly suggests that it signals via a BCMA-independent pathway and in an APRIL-dispensable way.

6/7/3 (Item 3 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16143164 BIOSIS NO.: 200100315003
Blocking of BAFF signaling pathway by BCMA-Fc attenuates autoimmune manifestations in BAFF transgenic mice and reveals its important role in maintaining peripheral B cell homeostasis

AUTHOR: Woodcock Stephen A (Reprint); Liu Zhong-Ying (Reprint);
Cachero
Teresa G (Reprint); Xian Fang (Reprint); Thill Greg (Reprint);
Ambrose
Christine (Reprint); Thompson Jeffery S (Reprint); Spinello Nicole
(Reprint); Mackay Fabienne; Browning Jeffrey L (Reprint); Kalled
Susan L
(Reprint); Wang LiChun (Reprint)
AUTHOR ADDRESS: Immunology and Inflammation, Biogen Inc., Cambridge,
MA,
USA**USA

JOURNAL: Blood 96 (11 Part 1): p616a November 16, 2000 2000
MEDIUM: print
CONFERENCE/MEETING: 42nd Annual Meeting of the American Society of
Hematology San Francisco, California, USA December 01-05, 2000;
20001201
SPONSOR: American Society of Hematology
ISSN: 0006-4971
DOCUMENT TYPE: Meeting; Meeting Abstract; Meeting Poster
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We have shown previously that the B cell
maturation antigen (BCMA) is a member of the TNF
receptor family and binds to B cell activating factor (BAFF)
expressed by
cells of dendritic and myeloid lineages. Treatment of normal mice
with a
soluble BCMA-Fc decoy, comprised of the extracellular domain
of human BCMA fused to the human IgG1 hinge, CH2 and CH3 domains,
resulted in the progressive loss of peripheral B cell subpopulations
including transitional(T)1, T2, mature and marginal zone cells. Bone
marrow hematopoiesis, however, was not affected. The reduction of
peripheral B cells by BCMA-Fc treatment is not Fc-dependent.
Additionally, treatment with BCMA-Fc in Baff transgenic mice
resulted in a reduction of elevated numbers of B cells,
splenomegaly, and
proteinuria, all hallmark disease phenotypes of BAFF transgenic
mice.
Taken together, the data provide in vivo evidence for the utility of
BCMA-Fc for treatment of diseases characterized by B cell
dysfunction.

6/7/4 (Item 4 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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16086684 BIOSIS NO.: 200100258523
TALL-1 is a target for B cell mediated autoimmune diseases in mice and
human
AUTHOR: Miner Kent T; Eastman Susan; Xia Xing Z; McCabe Susan; Hawkins
Nissa; Boone Tom; Delaney John; Lee Francis; Hsu Hailing; Khare
Sanjay D

JOURNAL: FASEB Journal 15 (5): pA1212 March 8, 2001 2001
MEDIUM: print
CONFERENCE/MEETING: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001 Orlando, Florida, USA March 31-April 04, 2001; 20010331
ISSN: 0892-6638
DOCUMENT TYPE: Meeting; Meeting Abstract
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: Members belonging to TNF superfamily are important in various aspects related to cell proliferation and death during immune regulation.

A recently discovered member of the TNF family, TALL-1 (Blys/BAFF/THANK/zTNF4) is involved in B cell proliferation and antibody production. Lupus prone NZBxNZWF1 mice at 2 months of age developed autoantibodies to ds-DNA and histone proteins when injected with recombinant soluble TALL-1 protein. Transgenic mice over expressing TALL-1 in non-lupus prone background showed increased antibody production and developed lupus like disease. TACI and BCMA have recently been identified as receptors for TALL-1. We examined a therapeutic effect of soluble receptors in lupus prone animals. Development of proteinuria (>300mg/dl) was delayed in both lupus prone MRL/lpr-lpr and NZBWF1 mice when treated with a recombinant soluble receptor fusion protein, TACI-Fc. TACI treatment also prolonged the survival time. We further examined serum levels of TALL-1 in several human autoimmune diseases. Patients with systemic lupus erythematosus, Myasthenia gravis and Wegener's granulomatosis showed significantly elevated levels of TALL-1 in serum when compared with healthy controls. Taken together, our data strongly suggest that TALL-1 is an important target in B cell mediated autoimmune diseases.

6/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15922018 BIOSIS NO.: 200100093857
APRIL and TALL-I and receptors BCMA and TACI: System for regulating humoral immunity
AUTHOR: Yu Gang; Boone Tom; Delaney John; Hawkins Nessa; Kelley Michael;

Ramakrishnan Meena; McCabe Susan; Qiu Wan-rong; Kornuc Masayo; Xia Xing-Zhong; Guo Jane; Stolina Marina; Boyle William J; Sarosi Ildiko; Hsu Hailing; Senaldi Giorgio; Theill Lars E (Reprint)
AUTHOR ADDRESS: Department of Inflammation, Amgen Inc., One Amgen Center Drive, Thousand Oaks, CA, 91320-1799, USA**USA
JOURNAL: Nature Immunology 1 (3): p252-256 September, 2000 2000
MEDIUM: print
ISSN: 1529-2908
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: We report that the tumor neurosis factor homolog APRIL (a proliferation-inducing ligand) stimulates in vitro proliferation of primary B and T cells and increases spleen weight due to accumulation of B cells in vivo. APRIL functions via binding to BCMA (B cell maturation antigen) and TACI (transmembrane activator and CAML-interactor) and competes with TALL-I (also called BlyS or BAFF) for receptor binding. Soluble BCMA and TACI specifically prevent binding of APRIL and block APRIL-stimulated proliferation of primary B cells. BCMA-Fc also inhibits production of antibodies against keyhole limpet hemocyanin and Pneumovax in mice, indicating that APRIL and/or TALL-I signaling via BCMA and/or TACI are required for generation of humoral immunity. Thus, APRIL-TALL-I and BCMA-TACI form a two ligands-two receptors pathway involved in stimulation of B and T cell function.

6/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15897239 BIOSIS NO.: 200100069078
A soluble form of B cell maturation antigen,
a receptor for the tumor necrosis factor family member APRIL,
inhibits tumor cell growth
AUTHOR: Rennert Paul; Schneider Pascal; Cachero Teresa G; Thompson Jeffrey;
Trabach Luciana; Hertig Sylvie; Holler Nils; Qian Fang; Mullen Colleen;
Strauch Kathy; Browning Jeffrey L; Ambrose Christine; Tschopp Jurg (Reprint)
AUTHOR ADDRESS: Institute of Biochemistry, University of Lausanne, Ch. des Boveresses 155, CH-1066, Epalinges, Switzerland**Switzerland

JOURNAL: Journal of Experimental Medicine 192 (11): p1677-1683
December 4,
2000 2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: A proliferation-inducing ligand (APRIL) is a ligand of the tumor necrosis factor (TNF) family that stimulates tumor cell growth in vitro and in vivo. Expression of APRIL is highly upregulated in many tumors including colon and prostate carcinomas. Here we identify B cell maturation antigen (BCMA) and transmembrane activator and calcium modulator and cyclophilin ligand (CAML) interactor (TACI), two predicted members of the TNF receptor family, as receptors for APRIL. APRIL binds BCMA with higher affinity than TACI. A soluble form of BCMA, which inhibits the proliferative activity of APRIL in vitro, decreases tumor cell proliferation in nude mice. Growth of HT29 colon carcinoma cells is blocked when mice are treated once per week with the soluble receptor. These results suggest an important role for APRIL in tumorigenesis and point towards a novel anticancer strategy.

6/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15720104 BIOSIS NO.: 200000438417
B cell maturation protein is a receptor for the tumor necrosis factor family member TALL-1
AUTHOR: Shu Hong-Bing (Reprint); Johnson Holly
AUTHOR ADDRESS: Department of Immunology, National Jewish Medical and Research Center and University of Colorado School of Medicine, 1400 Jackson Street, K516c, Denver, CO, 80206, USA**USA
JOURNAL: Proceedings of the National Academy of Sciences of the United States of America 97 (16): p9156-9161 August 1, 2000 2000
MEDIUM: print
ISSN: 0027-8424
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: TALL-1 is a recently identified member of the tumor necrosis

factor (TNF) family that costimulates B lymphocyte proliferation. Here we show that B cell maturation protein (BCMA), a member of the TNF receptor family that is expressed only by B lymphocytes, specifically binds to TALL-1. A soluble receptor containing the extracellular domain of BCMA blocks the binding of TALL-1 to its receptor on the plasma membrane and inhibits TALL-1-triggered B lymphocyte costimulation. Overexpression of BCMA activates NF-kappaB, and this activation is potentiated by TALL-1. Moreover, BCMA-mediated NF-kappaB activation is inhibited by dominant negative mutants of TNF receptor-associated factor 5 (TRAF5), TRAF6, NF-kappaB-inducing kinase (NIK), and IkappaB kinase (IKK). These data indicate that BCMA is a receptor for TALL-1 and BCMA activates NF-kappaB through a TRAF5-, TRAF6-, NIK-, and IKK-dependent pathway. The identification of BCMA as a NF-kappaB-activating receptor for TALL-1 suggests molecular targets for drug development against certain immunodeficient or autoimmune diseases.

6/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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15658954 BIOSIS NO.: 200000377267
BAFF binds to the tumor necrosis factor receptor-like molecule B cell maturation antigen and is important for maintaining the peripheral B cell population
AUTHOR: Thompson Jeffrey S; Schneider Pascal; Kalled Susan L; Wang LiChun; Lefevre Eric A; Cachero Teresa G; MacKay Fabienne; Bixler Sarah A; Zafari Mohammad; Liu Zhong-Ying; Woodcock Stephen A; Qian Fang; Batten Marcel; Madry Christine; Richard Yolande; Benjamin Christopher D; Browning Jeffrey L; Tsapis Andreas; Tschopp Jurg; Ambrose Christine (Reprint)
AUTHOR ADDRESS: Biogen, Inc., 12 Cambridge Center, Cambridge, MA, 02142, USA**USA
JOURNAL: Journal of Experimental Medicine 192 (1): p129-135 July 3, 2000
2000
MEDIUM: print
ISSN: 0022-1007
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: The tumor necrosis factor (TNF) family member B cell activating

factor (BAFF) binds B cells and enhances B cell receptor-triggered proliferation. We find that B cell maturation antigen (BCMA), a predicted member of the TNF receptor family expressed primarily in mature B cells, is a receptor for BAFF.

Although

BCMA was previously localized to the Golgi apparatus, BCMA was found to be expressed on the surface of transfected cells and tonsillar B cells. A soluble form of BCMA, which inhibited the binding of BAFF to a B cell line, induced a dramatic decrease in the number of peripheral B cells when administered in vivo. Moreover, culturing splenic cells in the presence of BAFF increased survival of a percentage of the B cells. These results are consistent with a role for

BAFF in maintaining homeostasis of the B cell population.

6/7/9 (Item 9 from file: 5)

DIALOG(R)File 5:Biosis Previews(R)

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15587774 BIOSIS NO.: 200000306087

TACI and BCMA are receptors for a TNF homologue implicated in B-cell autoimmune disease

AUTHOR: Gross Jane A (Reprint); Johnston Janet; Mudri Sherri; Enselman Rachel; Dillon Stacey R; Madden Karen; Xu Wenfeng; Parrish-Novak Julia;

Foster Don; Lofton-Day Cathy; Moore Margaret; Littau Alisa; Grossman Angelika; Haugen Harald; Foley Kevin; Blumberg Hal; Harrison Kim; Kindsvogel Wayne; Clegg Christopher H

AUTHOR ADDRESS: Department of Immunology, ZymoGenetics, 1201 Eastlake Avenue East, Seattle, WA, 98102, USA**USA

JOURNAL: Nature (London) 404 (6781): p995-999 April 27, 2000 2000

MEDIUM: print

ISSN: 0028-0836

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: B cells are important in the development of autoimmune disorders

by mechanisms involving dysregulated polyclonal B-cell activation, production of pathogenic antibodies, and co-stimulation of autoreactive T

cells. zTNF4 (BlyS, BAFF, TALL-1, THANK) is a member of the tumour necrosis factor (TNF) ligand family that is a potent co-activator of B

cells in vitro and in vivo. Here we identify two receptors for zTNF4 and

demonstrate a relationship between zTNF4 and autoimmune disease.

Transgenic animals overexpressing zTNF4 in lymphoid cells develop

symptoms characteristic of systemic lupus erythaematosus (SLE) and expand
a rare population of splenic B-1a lymphocytes. In addition,
circulating
zTNF4 is more abundant in NZBWF1 and MRL-lpr/lpr mice during the
onset
and progression of SLE. We have identified two TNF receptor family
members, TACI and BCMA, that bind zTNF4. Treatment of NZBWF1 mice
with soluble TACI-Ig fusion protein inhibits the development of
proteinuria and prolongs survival of the animals. These findings
demonstrate the involvement of zTNF4 and its receptors in the
development
of SLE and identify TACI-Ig as a promising treatment of autoimmune
disease in humans.

6/7/10 (Item 10 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)
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06131156 BIOSIS NO.: 198121015119
PRESENT ASPECTS OF LYMPHOCYTE ONTOGENESIS AND DIFFERENTIATION
AUTHOR: ASTALDI G (Reprint); TOPUZ U O; NERI A; IACOPINO P
AUTHOR ADDRESS: IST SIEROTERAPICO MILANESE S BELFANTI, VIA DARWIN,
20/22-
20143 MILANO**ITALY
JOURNAL: Bollettino dell'Istituto Sieroterapico Milanese 59 (4):
p255-292
1980
ISSN: 0021-2547
DOCUMENT TYPE: Article
RECORD TYPE: Citation
LANGUAGE: ITALIAN

6/7/11 (Item 1 from file: 24)
DIALOG(R)File 24:CSA Life Sciences Abstracts
(c) 2009 CSA. All rts. reserv.

0002291577 IP ACCESSION NO: 5313257
Polymorphism and chromosomal mapping of the mouse gene for B-cell
activating factor belonging to the tumor necrosis factor family
(Baff) and
association with the autoimmune phenotype

Jiang, Y; Ohtsuji, M; Abe, M; Li, N; Xiu, Y; Wen, XF; Shirai, T;
Hirose, S
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Hongo, Bunkyo-ku, Tokyo 113-8421, Japan

Immunogenetics, v 53, n 9, p 810-813, December 2001
PUBLICATION DATE: 2001

PUBLISHER: Springer-Verlag,
[URL:<http://link.springer.de/link/service/journals/00251/bibs/1053009/10530810.htm>]

DOCUMENT TYPE: Journal Article

RECORD TYPE: Abstract

LANGUAGE: English

ISSN: 0093-7711

DOI: 10.1007/s00251-001-0396-6

FILE SEGMENT: Genetics Abstracts; Immunology Abstracts

ABSTRACT:

B-cell activating factor (BAFF), also known as BlyS, THANK, and zTNF4, is a new member of the tumor necrosis factor (TNF) family that is constitutively produced by macrophages, dendritic cells, and activated T cells. BAFF is cleaved by furin protease, and the product is shed into the bloodstream. The cleaved BAFF product binds its receptor, TACI and BCMA specifically expressed on B cells and activates B-lineage cells. Elevated BAFF, as a result of injection or transgene expression, causes B-cell lymphadenopathy, CD5+ B1a cell expansion, plasmacytosis, or a systemic lupus erythematosus (SLE)-like autoimmune disease. These phenotypes appear to be due to up-regulation of antiapoptotic Bcl-2 expression in B cells, since B cells in BAFF-transgenic mice express high levels of Bcl-2. Relevant are findings that SLE-like disease occurs in mice expressing a bcl-2 transgene and in mice lacking the proapoptotic Bcl-2 family member Bim. SLE is a multifactorial autoimmune disease and much of the related pathology can be attributed to immune complexes formed by pathogenic high-affinity autoantibodies, including those against nuclear components. Genes that predispose to SLE are related to processes of emergence, activation, clonal expansion, differentiation, and maturation of autoreactive B cells. Such being the case, abnormalities in Baff may lead to the pathogenesis of SLE. (NZB x NZW)F1 mice are genetically susceptible to SLE and during aging spontaneously develop the disease. Gross and co-workers reported a strong correlation between disease progression and the amount of circulating serum BAFF (zTNF4) in (NZB x NZW)F1 mice. Because treatment of (NZB x NZW)F1 mice with soluble TACI-Ig fusion protein inhibited the development of immune complex-type glomerulonephritis (lupus

nephritis), and prolonged survival of the animals, BAFF may be a primary mediator of B cell-associated autoimmune disease in these mice. If this notion is tenable, the Baff allele of either NZB or NZW may be polymorphic.

6/7/12 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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09831500 Genuine Article#: 453UW Number of References: 20
Title: The role of TALL-1 and APRIL in immune regulation
Author(s): Khare SD (REPRINT) ; Hsu HL
Corporate Source: Amgen Inc, Dept Pathol Pharmacol, 1 Amgen Ctr Dr/Thousand Oaks//CA/91320 (REPRINT); Amgen Inc, Dept Pathol Pharmacol, Thousand Oaks//CA/91320; Amgen Inc, Dept Inflammation, Thousand Oaks//CA/91320
Journal: TRENDS IN IMMUNOLOGY, 2001, V22, N2 (FEB), P61-63
ISSN: 1471-4906 Publication date: 20010200
Publisher: ELSEVIER SCI LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, OXON, ENGLAND
Language: English Document Type: EDITORIAL MATERIAL
Abstract: Members of the tumor necrosis factor (TNF) superfamily play important roles in cell proliferation and death during immune regulation. Most members are synthesized as type II transmembrane proteins; the carboxy terminal extracellular domain can be cleaved from the cell membrane to form soluble active cytokines that bind to appropriate members of the TNF receptor family. Here, we describe the biological significance of recently discovered members of the TNF superfamily (TALL-1 and APRIL) and their receptors (TACI and BCMA) in the pathophysiology of human diseases.

6/7/13 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)
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133103739 CA: 133(8)103739s PATENT
Soluble receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins and their use in modulating the immune response and treating autoimmune disorders
INVENTOR(AUTHOR): Gross, Jane A.; Xu, Wenfeng; Madden, Karen; Yee, David
P.
LOCATION: USA
ASSIGNEE: Zymogenetics, Inc.
PATENT: PCT International ; WO 200040716 A2 DATE: 20000713

APPLICATION: WO 2000US396 (20000107) *US 226533 (19990107)

PAGES: 175 pp. CODEN: PIXXD2 LANGUAGE: English

PATENT CLASSIFICATIONS:

CLASS: C12N-015/11A; C07K-014/705B; A61K-038/17B; A61K-039/395B

DESIGNATED COUNTRIES: AE; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY;

CA; CH;

CN; CR; CU; CZ; DE; DK; DM; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU;

ID; IL;

IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU; LV; MD; MG;

MK; MN;

MW; MX; NO; NZ; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TR;

TT; UA;

UG; UZ; VN; YU; ZA; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM

DESIGNATED REGIONAL: GH; GM; KE; LS; MW; SD; SL; SZ; TZ; UG; ZW;

AT; BE;

CH; CY; DE; DK; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; BF;

BJ; CF;

CG; CI; CM; GA; GN; GW; ML; MR; NE; SN; TD; TG

SECTION:

CA215010 Immunochemistry

IDENTIFIERS: TACI isoform BR43x2 immune modulation autoimmune disorder,

sequence BR43x2 protein cDNA human mouse, BCMA protein immune modulation autoimmune disorder

DESCRIPTORS:

Proteins,specific or class...

BCMA (B cell membrane antigen); soluble receptor BR43x2 isoform of transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating autoimmune disorders...

bronchitis, treatment of; soluble receptor BR43x2 isoform of transmembrane

activator and CAML-interactor TACI and related proteins and their use

in modulating the immune response and treating autoimmune

Proteins,specific or class...

BR43x2; soluble receptor BR43x2 isoform of transmembrane activator and

CAML-interactor TACI and related proteins and their use in modulating

the immune response and treating autoimmune disorders

Proteins,specific or class...

CAML (calcium-modulator and cyclophilin ligand); soluble receptor BR43x2

isoform of transmembrane activator and CAML-interactor TACI and related

proteins and their use in modulating the immune response a

Intestine...

colon, expression specificity in; soluble receptor BR43x2 isoform of

transmembrane activator and CAML-interactor TACI and related proteins

and their use in modulating the immune response and treating aut
Intestine,disease...
Crohn's, treatment of; soluble receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Joint,anatomical...
disease, treatment of; soluble receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune dis
Appendix... B cell(lymphocyte)... Bone marrow... Lung... Lymph node...
Lymphoma... Salivary gland... Spleen... Stomach... Testis...
Trachea(anatomical)...
expression specificity in; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating
autoimmune
Immunoglobulins...
fusion products; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune
disorders
Transplant and Transplantation...
graft-vs.-host reaction, treatment of; soluble receptor BR43x2
isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treatin
Antibodies...
humanized; soluble receptor BR43x2 isoform of transmembrane
activator and
CAML-interactor TACI and related proteins and their use in
modulating
the immune response and treating autoimmune disorders
Diabetes mellitus...
insulin-dependent, treatment of; soluble receptor BR43x2 isoform
of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating auto
Tumor necrosis factors...
ligand neutrokin α ; soluble receptor BR43x2 isoform of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating
autoimmune
Antibodies...

monoclonal; soluble receptor BR43x2 isoform of transmembrane
activator and
CAML-interactor TACI and related proteins and their use in
modulating
the immune response and treating autoimmune disorders
Nerve,disease...
neuropathy, Ig light chain, treatment of; soluble receptor BR43x2
isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and trea
Cytokines...
neutrokin α ; soluble receptor BR43x2 isoform of transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune disord
Lymphoma...
non-Hodgkin's, expression specificity in; soluble receptor BR43x2
isoform
of transmembrane activator and CAML-interactor TACI and related
proteins and their use in modulating the immune response and trea
Salivary gland...
parotid, neoplasm, expression specificity in; soluble receptor
BR43x2
isoform of transmembrane activator and CAML-interactor TACI and
related
proteins and their use in modulating the immune response and
Animal cell line...
Raji, expression specificity in; soluble receptor BR43x2 isoform
of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating auto
Shock(circulatory collapse)...
septic, treatment of; soluble receptor BR43x2 isoform of
transmembrane
activator and CAML-interactor TACI and related proteins and their
use
in modulating the immune response and treating autoimmune diso
Intestine...
small, expression specificity in; soluble receptor BR43x2 isoform
of
transmembrane activator and CAML-interactor TACI and related
proteins
and their use in modulating the immune response and treating aut
Antibodies... cDNA sequences... Drugs... Gene therapy...
Immunosuppression
... Mammal(Mammalia)... Molecular cloning... Mouse... Primate...
Protein
sequences...
soluble receptor BR43x2 isoform of transmembrane activator and
CAML-interactor TACI and related proteins and their use in
modulating

the immune response and treating autoimmune disorders
 Lupus erythematosus...
 systemic, treatment of; soluble receptor BR43x2 isoform of
 transmembrane
 activator and CAML-interactor TACI and related proteins and their
 use
 in modulating the immune response and treating autoimmune di
 Proteins,specific or class...
 TACI (transmembrane activator and CAML-interactor); soluble
 receptor
 BR43x2 isoform of transmembrane activator and CAML-interactor
 TACI and
 related proteins and their use in modulating the immune respons
 Amyloidosis... Anemia(disease)... Asthma... Autoimmune disease...
 Emphysema
 ... Inflammation... Kidney,disease... Kidney,neoplasm... Multiple
 myeloma
 ... Multiple sclerosis... Myasthenia gravis... Rheumatoid arthritis...
 Swelling,biological... Transplant rejection...
 treatment of; soluble receptor BR43x2 isoform of transmembrane
 activator
 and CAML-interactor TACI and related proteins and their use in
 modulating the immune response and treating autoimmune disorders
 Fusion proteins(chimeric proteins)...
 with Ig heavy chain; soluble receptor BR43x2 isoform of
 transmembrane
 activator and CAML-interactor TACI and related proteins and their
 use
 in modulating the immune response and treating autoimmune disor
 CAS REGISTRY NUMBERS:
 217638-65-8 282738-49-2 amino acid sequence; soluble receptor
 BR43x2 isoform
 of transmembrane activator and CAML-interactor TACI and related
 proteins and their use in modulating the immune response and
 treating
 autoimmune disorders
 283157-99-3 cysteine-rich pseudo repeat domain; soluble receptor
 BR43x2
 isoform of transmembrane activator and CAML-interactor TACI and
 related
 proteins and their use in modulating the immune response and
 treating
 autoimmune disorders
 156253-82-6 198123-04-5 282738-43-6 282738-45-8 282738-47-0
 282738-48-1 nucleotide sequence; soluble receptor BR43x2 isoform
 of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 148997-64-2D 198029-64-0D 282738-44-7D 282738-46-9D subfragments
 are

claimed, amino acid sequence; soluble receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 225456-59-7 271755-95-4 274950-83-3 274950-84-4 282738-77-6
 282738-78-7 282738-79-8 282738-80-1 282738-81-2 282738-82-3
 282738-83-4 282738-84-5 282738-85-6 282738-86-7 282738-87-8
 282738-88-9 282738-89-0 282738-90-3 282738-91-4 282738-92-5
 282738-93-6 282738-94-7 282738-95-8 282738-96-9 282738-97-0
 282738-98-1 282738-99-2 282739-00-8 282739-02-0 282739-03-1
 282739-04-2 282739-05-3 282739-06-4 282739-07-5 282739-08-6
 282739-09-7 282739-10-0 282739-11-1 282739-12-2 282739-13-3
 unclaimed nucleotide sequence; soluble receptor BR43x2 isoform of
 transmembrane activator and CAML-interactor TACI and related
 proteins
 and their use in modulating the immune response and treating
 autoimmune
 disorders
 282738-76-5 282739-01-9 unclaimed protein sequence; soluble
 receptor BR43x2
 isoform of transmembrane activator and CAML-interactor TACI and
 related
 proteins and their use in modulating the immune response and
 treating
 autoimmune disorders
 98849-88-8 256922-04-0 282729-02-6 282729-03-7 unclaimed
 sequence; soluble
 receptor BR43x2 isoform of transmembrane activator and
 CAML-interactor
 TACI and related proteins and their use in modulating the immune
 response and treating autoimmune disorders
 ? ds

Set	Items	Description
S1	62	TRANSMEMBRANE AND DOMAIN AND (BCMA OR B CELL
MATURATION AN-		
TIGEN)		
S2	17	RD S1 (unique items)
S3	1744	BCMA OR (B(W) CELL(W) MATURATION (W) ANTIGEN)
S4	280	SOLUBLE AND S3
S5	73	RD S4 (unique items)
S6	13	S5 NOT PY>2001
? logoff y		
06apr09 09:36:39 User226352 Session D1127.3		
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	\$41.48	17 Type(s) in Format 7
	\$41.48	17 Types
\$48.57	Estimated cost File5	
	\$0.47	0.062 DialUnits File6
\$0.47	Estimated cost File6	

	\$2.19	0.340	DialUnits	File24
	\$2.70	1	Type(s)	in Format 7
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\$0.28	Estimated cost		File41	
	\$1.07	0.207	DialUnits	File45
\$1.07	Estimated cost		File45	
	\$0.86	0.181	DialUnits	File50
\$0.86	Estimated cost		File50	
	\$1.37	0.322	DialUnits	File65
\$1.37	Estimated cost		File65	
	\$5.57	0.512	DialUnits	File71
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	\$17.24	1.245	DialUnits	File72
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	\$7.66	2	Types	
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	\$3.57	1.013	DialUnits	File155
\$3.57	Estimated cost		File155	
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\$1.42	Estimated cost		File156	
	\$0.43	0.092	DialUnits	File162
\$0.43	Estimated cost		File162	
	\$1.02	0.074	DialUnits	File172
\$1.02	Estimated cost		File172	

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$0.57 Estimated cost File305
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    $0.10    0.028 DialUnits File370
$0.10 Estimated cost File370
    $0.21    0.071 DialUnits File393
$0.21 Estimated cost File393
    $14.53   1.112 DialUnits File399
        $5.96 2 Type(s) in Format 7
        $5.96 2 Types
$20.49 Estimated cost File399
    $1.77    0.062 DialUnits File434
$1.77 Estimated cost File434
    OneSearch, 29 files, 10.773 DialUnits FileOS
    $4.53 TELNET
$211.62 Estimated cost this search
$211.64 Estimated total session cost 11.157 DialUnits
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